Foreign Animal Disease Report

United States Department of Agriculture
Animal And Plant Health Inspection Service
Veterinary Services

Emergency Programs

Number 20-1

Spring 1992

This Issue

Emergency Programs Activities

Field Investigations. During the first quarter of fiscal year (FY) 1992 (October 1–December 31, 1991), veterinarians from the U.S. Department of Agriculture (USDA), Animal and Plant Health Inspection Service (APHIS), Veterinary Services (VS), and State departments of agriculture conducted 27 investigations of suspicious foreign animal diseases in the United States and Puerto Rico to eliminate the possibility that an exotic disease may have been introduced. These investigations included 11 for vesicular conditions, 2 for swine septicemic conditions, 1 for mucosal conditions, 6 for exotic Newcastle disease in pet birds and poultry, 3 for encephalitic conditions, and 4 for undesignated conditions.

There were 5 investigations in 8 States in the Northern Region, 13 investigations in 8 States in the Southeastern Region, 4 investigations in 3 States in the Central Region, and 5 investigations in 4 States in the Western Region. No foreign animal diseases or pests were found.

Health Concerns for Captive-Raised Waterfowl. On December 11, 1991, the APHIS, VS, Emergency Programs staff participated in a tri-State agency meeting to discuss health concerns about captive-raised wild waterfowl. Topics included the potential effect of diseases in captive-raised birds on native waterfowl populations, regional emergency preparedness in the event of a disease outbreak, and the feasibility of a health certification program for captive-raised waterfowl. State agriculture and wildlife agencies from Delaware, Maryland, and Virginia; Federal entities such as the U.S. Department of the Interior and USDA; and the Southeastern Cooperative Wildlife Disease Study were represented, along with the poultry industry.

Emergency Preparedness. During February 4–6, 1992, a workshop was held in Hyattsville, MD, to review and revise the Regional Emergency Animal Disease Eradication Organization (READEO) structure. This review included evaluating (1) the functional responsibilities of each position in the READEO and (2) the information-generating capability of the Recorded Emergency Animal Disease Information (READI) System. The directors and the administrative staff, field operations staff, and

technical support officers of all four READEO's participated. These directors and officers will build on the decisions reached at the workshop to develop common procedures for operations applicable to all four READEO's. Later, the READEO manual will be rewritten.

Training Activities for the Third and Fourth Quarters of FY 1992. Training courses designed to enhance foreign animal disease detection and emergency response have been planned and schedules have been distributed within APHIS and to State cooperators. Training planned for the third quarter includes

Foreign Animal Disease Seminar, April 7–9, 1992, Southeastern Region

READEO Workshop: April 14–16, 1992, Northern Region; April 28–30, Western Region; June 8–12, Southeastern Region

Foreign Animal Disease Awareness for Animal Health Technicians: April 21–23, Northern Region; June 2–4, Central Region

Foreign Animal Disease Diagnostician's Course, May 4–15, at the National Veterinary Services Laboratories (Ames, IA) and the Plum Island Animal Disease Diagnostic Laboratory (Plum Island, NY)

Training planned for the fourth quarter of 1992 includes

Foreign Animal Disease Threats and Implications, July 7–10, in College Park, MD

Wildlife Diseases Seminar for Foreign Animal Disease Diagnosticians, August 18–21, Athens, GA

(Dr. M. A. Mixson, Emergency Programs, VS, APHIS, USDA, Hyattsville, MD 20782, 301-436-8073)

Foreign Animal Disease Update

This update consolidates information from Office International des Epizooties (OIE) bulletins into tables covering August and September 1991. Countries reporting disease outbreaks are listed below the appropriate disease heading (followed by the month/year of the report and total number of outbreaks reported for that time period). The notation "+" indicates that the presence of disease was reported without information on total number of outbreaks. Outbreak number followed by "+" indicates number of outbreaks as well as disease presence.

Foot-and-Mouth Disease

Virus Untyped
Chad (7/91) +
Argentina (5&6/91) 40
Pakistan (7&8/91) 2+
Myanmar (7/91) 2
Paraguay (8&9/91) 11
Bhutan (8/91) +
Hong Kong (6–8/91) 8

Virus O
Morocco (9/91) 2
Georgia (9&8/91) 11
Argentina (5&6/91) 6
Colombia (6&7/91) 4
Oman (5&6/91) 109
Pakistan (8&9/91) +
Turkey (7/91) 100
Paraguay (8&9/91) 10
Nepal (2,6,8,&9/91) +
Kyrgyzstan (9/91) 2

Virus A
Argentina (5&6/91) 13
Colombia (6–8/91) 19
Pakistan (7&8/91) +
Turkey (7/91) 23
Kenya (5/91) 2
Ecuador (1–7/91) 2
Turkey (8/91) 11

Virus C
Bhutan (8/91) +
Argentina (5/91) 1

Virus SAT 2
Mali (6–9/91) 13
Kenya (8/91) 1

Virus SAT 3
Zimbabwe (7/91) 1

Virus Asia 1
Pakistan (7&8/91) +

Vesicular Stomatitis

Virus Unknown
Panama (7&8/91) 3
Mexico (7/91) 1

Virus Indiana
Colombia (6–7/91) 28

Virus New Jersey
Panama (7/91) 1
Colombia (6–8/91) 63
Costa Rica (6&7/91) 4
El Salvador (5–7/91) 12
Guatemala (6/91) 1
Honduras (7/91) 4
Nicaragua (7/91) 2
Ecuador (3/91) 1

Swine Vesicular Disease
Italy (4/91) 2

Rinderpest
*Mongolia (8/91) 3
Ethiopia (9/91) 2

Peste des Petits Ruminants
Senegal (6&7/91) 2
Oman (5&6/91) 23
Guinea (7&8/91) +

Contagious Bovine Pleuropneumonia
Italy (8&9/91) 10
Portugal (5–7/91) 381
Guinea (7–9/91) +
Mali (7&8/91) 4
Kenya (5&8/91) 14

Lumpy Skin Disease
Senegal (6&7/91) 2
Botswana (7/91) +
Zimbabwe (7&8/91) 18
Kenya (7/91) 2
South Africa (8/91) +

Rift Valley Fever
Mozambique (8/91) +

Bluetongue
United States (8&9/91) +
Malaysia (1–6/91) +
Israel (9/91) 1

Sheep and Goat Pox
Senegal (6&7/91) 5
Oman (5&6/91) 12
Turkey (7&8/91) 72

African Horse Sickness
Morocco (8&9/91) 33
Mozambique (8/91) +
Senegal (6&7/91) 2+
Zimbabwe (7/91) 2
South Africa (8/91) +

* This is the first sign of rinderpest in Mongolia since 1935.

African Swine Fever
Mozambique (8&9/91) 1+
Italy (8&9/91) 12
Senegal (7/91) 1
Portugal (7&8/91) 11
Spain (8&9/91) 35

Hog Cholera
Czechoslovakia (7&8/91) 4
Russia (9/91) 9
Mexico (7&8/91) 9
Argentina (5&6/91) +
Colombia (6&8/91) 6
Malaysia (1–6/91) +
Taiwan (7&8/91) 17
Yugoslavia (6/91) 9
Paraguay (8&9/91) 3
Italy (9/91) 1
U.S.S.R. (Savropol) (8/91) 1
Ecuador (8/91) 1
Korea(s) (8/91) 2
Hong Kong (6/91) 2
Austria (8/91) 2

Newcastle Disease
Mozambique (8/91) +
Portugal (8/91) 5
Egypt (7&8/91) 6
Senegal (7/91) +
Mexico (7&8/91) 8
Colombia (6/91) +
Albania (6/91) 1
Yugoslavia (7/91) 3
Turkey (7&8/91) 4
Guinea (7&8/91) +
South Africa (8/91) 2
Ecuador (1–8/91) +
Hong Kong (6/91) 5
Japan (5/91) 4

Velogenic Viscerotropic Newcastle Disease
Mexico (7/91) 1
South Korea (7&8/91) 8
Malaysia (Penin) (1–6/91) +
Myanmar (7/91) 1
Kenya (4,5,7,&8/91) 11

Bovine Spongiform Encephalopathy
Switzerland (8/91) 2

Scrapie
Czechoslovakia (7&8/91) 5
Norway (5/91) 1

Porcine Reproductive and Respiratory Syndrome
Belgium (3–6/91) 75

(Dr. Peter Fernandez, International Services, APHIS, USDA, Hyattsville, MD 20782, 301-436-8892)

A Serologic Occurrence of Viral Turkey Rhinotracheitis

A North Carolina turkey hen farm experienced a sharp drop in egg production in the fall of 1991. (Over a period of 1 week, the percentage of hens laying eggs decreased from 65 to 20.) Diagnostic testing done in October 1991 revealed positive serology for viral turkey rhinotracheitis (VTRT). This was the first time VTRT antibodies have been found in the United States. Attempts to isolate a virus were unsuccessful.

The affected turkey farm is located in southeast Anson County, near Morven, NC. The farm is a commercial turkey egg-production facility with four turkey barns plus an office building. Each barn contains about 3,500 breeder turkey hens. The barns are maintained as individual units and are aligned adjacent to each other with the doorways facing in an east–west direction. The office and the gate entrances are on the west side facing a county road. The turkey hens were artificially inseminated once a week by the farm crew. Feed delivery was also made once a week. The biosecurity on the farm is excellent. Only the two center houses tested positive on serology, and no virus could be isolated. There were no clinical signs typical of VTRT in either of the houses; the only symptom was the drop in egg production. Furthermore, only a low percentage (less than 13 percent) of the flock had positive titers; this does not fit the clinical pattern as seen in other countries with VTRT. Surveillance of other poultry flocks in the area and progeny testing showed no evidence of spread to other flocks or any egg-associated transmission. There has been no clinical evidence of VTRT in the Carolinas.

(Dr. Adam Grow, Emergency Programs, VS, APHIS, USDA, Hyattsville, MD 20782, 301-436-8073)

Bovine Spongiform Encephalopathy Update

Bovine spongiform encephalopathy (BSE) is a fatal degenerative disease affecting the central nervous system of cattle. The condition was first officially diagnosed in Great Britain in 1986, and, at press time, had not been reported in the United States. As of February 7, 1992, the British Ministry of Agriculture, Fisheries and Foods reported 47,616 head of cattle in 16,148 herds confirmed to have BSE in the United Kingdom. Additional cases were then occurring at the rate of approximately 1,000 per week(1). There were 6,055 brain samples pending diagnosis at that time. The disease has also been confirmed in Ireland, Oman (2 imports), the Falkland Islands (1 import), Switzerland (10 domestic cases), and France (5 domestic cases).

BSE belongs to a group of related diseases known as the transmissible spongiform encephalopathies. This family includes scrapie (which affects sheep and goats), transmissible mink encephalopathy, and chronic wasting disease of mule deer and elk. Humans may be affected by three like diseases: kuru, Creutzfeldt–Jakob disease, and Gerstmann–Straussler syndrome. These diseases are caused by an as-yet uncharacterized agent that produces spongiform changes in the brain. These encephalopathies are typified by a long incubation period, insidious development of clinical signs (which include motor abnormalities and behavioral changes), and the lack of any detectable host immune or inflammatory response.

The causative agent is extremely resistant to heat and most other sterilization processes. The nature of the etiologic agent has been described as (1) an unconventional virus, (2) a prion (defined as "a proteinaceous infectious particle that resists inactivation and which modifies nucleic acids and contains an abnormal isoform of a cellular protein"), and (3) a virino (an agent genome, most likely a nucleic acid, protected by a host protein).

Great Britain's Situation

BSE was first diagnosed by the British Ministry of Agriculture, Fisheries and Foods' Central Veterinary Laboratory at Weybridge, England, in November 1986. However, it is thought that the disease may have first surfaced as early as April 1985. The majority of cases in Great Britain have been in Friesian dairy cattle between 3 and 5 years of age. The ages of afflicted cows range from 22 months to 17 years. The confirmed total herd incidence in the United Kingdom is 16.9 percent. The dairy herd incidence is 30.84 percent, and beef herd incidence is 4.04 percent (February 7, 1992, figures).

Epidemiologic data indicate that BSE in Great Britain was caused by feeding cattle meat and bone meal produced from the rendered carcasses of scrapie-infected sheep. The practice of using meat and bone meal as a protein source in cattle rations has been common for several decades in Great Britain. But initial epidemiologic studies identified changes in rendering operations during the early 1980's. These included a switch from batch processing to continuous processing, and the discontinuation of hydrocarbon solvent extraction of fat from meat and bone meal(2). The current thinking is that the 1980's rendering process allowed a substantial dose of the scrapie agent to survive and infect the feeding cattle. Infected cattle were then recycled (via rendering) through the food chain, thus amplifying the number of new cases. Another theory suggests that there was an existing, low-level spongiform encephalopathy in the cattle population that was increased by feeding rendered cattle carcasses back to cattle(3). This second theory excludes the involvement of sheep in the development of the disease.

In the rendering process, hydrocarbon solvent extraction of fat involves two applications of heat. The second heat treatment uses moist heat similar to that of an autoclave. Autoclaving is known to aid in lowering the infectivity of the scrapie agent(4). Further investigation by Wilesmith and others(5) does not support the theory that the introduction of the continuous rendering process was primarily responsible for the exposure of cattle to a scrapielike agent. It does suggest that eliminating the agent's exposure to the organic solvent and to the moist heat treatment may have allowed higher titers of the agent to remain in the meat and bone meal. These titers would then be sufficient to infect bovines.

There have been 16 cases of BSE reported in cattle born since the implementation of the July 1988 regulation that prohibits feeding ruminant proteins to ruminants. The first affected animal was a 26-month-old Guernsey cow that had no history of receiving feed with a ruminant-based protein in it. The animal's dam was previously confirmed to have had BSE. There is suspicion that the other 15 infected cows may have received ruminant protein from feed not disposed of after the ban went into effect. Although these cases may point to the possibility of maternal transmission, there is not enough evidence to draw any conclusions.

As of April 1992, there is still no evidence that BSE is transmitted by sheep-to-cattle contact. In Great Britain, BSE has been experimentally transmitted to cattle, mice, sheep, and a pig by intracranial injection. It has also been transmitted orally to mice.

Cattle affected by BSE experience a progressive nervous system degeneration. This may cause the animal to display changes in temperament, such as nervousness or aggression; abnormal posture, incoordination, and difficulty in rising; decreased milk production; and loss of body weight despite continued appetite. All affected cattle eventually die. The incubation period is thought to be from 2 to 8 years with the clinical course of the disease lasting from 2 weeks to 6 months. There is no treatment.

The diagnosis of BSE is made by the observation of clinical signs. Confirmation is by postmortem histologic examination of the brain. There is no test to detect the disease in live animals.

To date, no scientific evidence indicates that BSE is a human health hazard.

There have been other manifestations of spongiform encephalopathies in Great Britain. These have occurred in cats, kudus, an eland, a nyala, and a gemsbok.

As a result of BSE, Britain has taken a number of actions, including:

1. Prohibiting the inclusion of ruminant-derived protein in all ruminant rations. Because investigators were able to transmit BSE to a pig, specified offal from all bovines (brain, spinal cord, spleen, thymus, and intestines with the mesenteric lymph nodes) can no longer be fed to warmblooded animals.

2. Prohibiting the consumption of milk and meat from affected cattle by either animals or humans.

3. Destroying all animals displaying clinical signs of BSE and indemnifying the owners.

USDA Actions

1. APHIS has prohibited the importation of ruminants from the United Kingdom since July 1989.

2. In November 1989, APHIS banned the importation of fetal bovine serum from the United Kingdom. There has also been a ban placed on other ruminant-origin products from countries known to have BSE. These products include meat-and-bone meal, bone meal, blood meal, offal, fat, and glands. The official regulation went into effect December 6, 1991.

In addition to prohibiting the materials listed above, the regulation requires that imported meat from the ruminants in the Bovidae family be deboned with visible lymphatic and nervous tissue removed, that it be obtained from animals which have undergone a veterinary examination prior to slaughter, and that it be obtained from ruminants which have not been in any country affected with BSE during a period of time when the country permitted the use of ruminant protein in ruminant feeds. This was not done because of a human health risk but to prevent potentially contaminated ruminant trimmings from entering the animal food chain in the United States.

Other ruminant products either are prohibited or must be imported under permit for scientific, educational, research, or cosmetic purposes.

3. Surveillance efforts have been stepped up in the United States to verify America's BSE-free status and to detect the disease promptly should it be introduced. The surveillance efforts include:

   a. Examination of brains from cattle over 2 years of age exhibiting neurologic signs. More than 60 laboratories throughout the country have agreed to forward tissue to the National Veterinary Services Laboratories in Ames, IA, for histologic examination. APHIS is working in cooperation with Iowa State University to conduct this project. Two pathologists have gone to England for training. To date, they have examined 103 brains.

      The Centers for Disease Control are screening rabies-negative cattle brains from public health laboratories. That agency has examined 135 brains as of February 3, 1992.

   b. Field investigations of suspicious disease conditions by more than 230 APHIS and State veterinarians with special training in diagnosing foreign animal diseases.

   c. The distribution of educational materials (including factsheets, an informational packet, and a British videotape) to industry groups, veterinarians in private practice, colleges of veterinary medicine, APHIS field offices, and State- and extension-employed veterinarians. This effort is being made to inform individuals about the disease and to increase their awareness of its signs.

   d. The coordination of activities with other agencies, such as the USDA Food Safety and Inspection Service, to examine downer cows at slaughter and the Centers for Disease Control to examine brains from rabies-negative but BSE-symptomatic cattle.

4. Scientific research. USDA's Agricultural Research Service (ARS) has allocated slightly over a million dollars for scrapie/BSE research in FY 1992. ARS has projects on the molecular composition of the agent, efforts for a preclinical test for scrapie, and rendering studies to determine if the scrapie agent will survive the rendering process used in the United States and cause disease in laboratory animals or cattle.

5. Tracing imported cows. APHIS is tracing and monitoring cattle imported between 1981 and the ban on importation of ruminants from the United Kingdom. As of December 10, 1991, of the 459 head traced, 393 have been located and found to be free of signs suggestive of BSE.

6. APHIS established a BSE Issue Management Committee to advise agency management on all aspects of BSE policy. This committee compiled a report(6) that examined seven major areas of concern:

    a. Comparative risk. This study looked at animal populations, production practices, rendering practices, and relevant regulations in the United States and the United Kingdom to determine if significant differences in BSE risk existed between the two countries. The findings suggest that the United States has very different risk factors than those found in the United Kingdom.

    b. Rendering policy.

    c. Contingency plans in the event BSE were to be confirmed in the United States.

    d. Surveillance. The committee examined what efforts are being made at present and what could be done to further enhance surveillance for BSE.

    e. Public relations. The committee evaluated the problems experienced with the media in the United Kingdom and analyzed strategies and options for managing APHIS' public relations in regard to BSE.

    f. Research.

    g. The impact of BSE on the APHIS scrapie program.

7. APHIS officials continue to meet with other governmental agencies and industry groups to exchange information and policy ideas about BSE.

Note: It is **imperative** that U.S. veterinarians and livestock producers maintain awareness about BSE and its clinical signs and submit any animals with suspect signs for diagnostic purposes. There is suspicion throughout the world about the United States' apparent freedom from BSE. Hence, to ensure early detection and sustain U.S. export markets, it is necessary to actively seek a diagnosis of any potential case of the disease.

References

1. Written communication from Dr. John W. Wilesmith, Epidemiology Unit, Central Veterinary Laboratory, Weybridge, England, February 7, 1992.

2. Wilesmith, J. W.; Wells, G. A.; Cranwell, M. P.; Ryan, J.B M. 1988. Bovine spongiform encephalopathy: epidemiological studies. Veterinary Record 123: 638–644.

3. Personal communication with J. W. Wilesmith, November 1990.

4. Brown, P.; Rohwer, R. G.; Gajdusek, D. C. 1986. Newer data on the inactivation of scrapie virus or Creutzfeldt–Jakob disease virus in brain tissue. Journal of Infectious Diseases 153(6): 1145–1148.

5. Wilesmith, J. W.; Ryan, J.B.M.; Atkins, M. J. 1991. Bovine spongiform encephalopathy: epidemiological studies on the origin. Veterinary Record 128(9): 199–203.

6. USDA, APHIS, VS. 1991. Qualitative analysis of bovine spongiform encephalopathy risk factors in the United States. Hyattsville, MD: U.S. Department of Agriculture, Animal and Plant Health Inspection Service, Veterinary Services.

(Dr. Linda A. Detwiler, VS, APHIS, USDA, Trenton, NJ 08619, 609-989-2255)

New Methods for Diagnosis of Malignant Catarrhal Fever

Malignant catarrhal fever (MCF) is most typically a sporadic (but occasionally epizootic), acute syndrome in cattle and several other ruminant species that is frequently fatal. The disease is characterized by intense oronasal and conjunctival inflammation, corneal opacity, and multisystemic lymphocellular proliferation with fibrinoid and/or mononuclear cellular vasculitis. Infection usually occurs following contact with infected wildebeest, other antelopes, or (especially in Europe and the United States) sheep. In most cases, the carrier animals have an inapparent infection. Recently, the etiologic agent of MCF associated with wildebeest contact has been placed into a new herpesvirus group, the gammaherpesviruses, and has been named alcelaphine herpesvirus-1 (AHV-1). A gammaherpesvirus strain isolated from other species of African antelope has been named AHV-2. This gammaherpesvirus has not yet been associated with disease in cattle but, like AHV-1, produces inapparent infection in reservoir ruminant species. The two viruses appear to differ in their animal reservoirs and cell culture specificities. In cattle, MCF usually is presumed to be caused by a third strain of virus, called "sheep-associated MCF virus," which until recently had never been grown in cell culture.

Confirmatory viral diagnosis of MCF is tedious and often unrewarding. Specimen autolysis or rapid freezing eliminates viral infectivity. Further, unless they are from neonatal or juvenile animals, the infected tissues contain only highly cell-associated virus that replicates slowly and may have to undergo repeated cell passages before cytopathic effect is seen. Passage procedures appropriate to isolation of cell-associated viruses are particularly stringent. The virus can be passed only within living cells that, if frozen, must be kept viable with chemical cryoprotectants. These viruses also are hard to demonstrate by fluorescent antibody staining and electron microscopy. Because of these difficulties in isolating or visualizing the virus, serology has always been the primary diagnostic technique used.

The basis for the high sensitivity of the polymerase chain reaction (PCR) is that only one or, at most, several viral particles are necessary for amplification to detectable levels, and the virus need not be viable. Short sequences of viral DNA in buffy coat cells are recognized by complementary primer sequences prepared from known viral DNA. By alternately heating and cooling the mixture of primer and virus in the presence of polymerizing enzyme, geometrically increasing amounts of primer-specific DNA are formed in samples positive for MCF. The presence of amplified DNA can then be confirmed by using other enzymes that break it down so that the component parts can be compared to similar (restriction endonuclease) products of known MCF virus. To increase the sensitivity and specificity of the procedure, two nested primers frequently are chosen for a two-stage PCR.

Highly sensitive PCR procedures were already in use for viral diagnosis at the National Veterinary Services Laboratories, and use of such a procedure for diagnosis of MCF appeared to be a logical approach. The senior author, in cooperation with scientists at the National Animal Disease Center, undertook development of a two-stage test with nested primers. This work was reported in a recent publication (Katz, J.; Seal, B.; Ridpath, J. 1991. Molecular diagnosis of alcelaphine herpesvirus [malignant catarrhal fever] infections by nested amplifications of viral DNA in bovine buffy coat specimens. Journal of Veterinary Diagnostic Investigations 3: 193–198.) Experimentally it was possible to identify AHV-1 and AHV-2 viruses in bovine blood and cell culture origin specimens. Each of five AHV-1 and two AHV-2 isolates was specifically positive by PCR, while bovine herpesvirus-1 (infectious bovine rhinotracheitis), bovine herpesvirus-2 (bovine herpes mammillitis), and bovine herpesvirus-4 (DN-599) were PCR negative. In experimental animal infections, four calves were inoculated with AHV-1. Three remained both serologically and PCR negative. In the fourth calf, viral DNA was detected in vivo only 3 days postinfection, whereas seroconversion first occurred 14 days postinfection. Viral DNA was also detected in a 10^8 dilution of 10^6 $TCID_{90}$/mL reference virus, attesting to the sensitivity of the technique. Noninfectious, defective, interfering viral particles, and/or the possibility that the PCR target may be a repeated sequence may account for the detection of as little as 0.01 $TCID_{50}$ of AHV-1.

MCF is a disease that usually is not easily or rapidly diagnosed by conventional virus isolation methods. The PCR and associated procedures described herein may substantially enhance the virologic diagnosis of clinically overt, persistent subclinical, and latent AHV-1 and AHV-2 infections. A herpesvirus recently was isolated for the first time from sheep-associated MCF (Schuller, W., et al. 1990. Evidence that the sheep-associated form of malignant catarrhal fever is caused by a herpesvirus. Journal of Veterinary Medicine 37: 442–447). Although it is serologically related to AHV-1, the reactivity of that virus in the test described here is as yet unknown.

The PCR method currently utilized depends on demonstration of virus in buffy coat cells, theoretically making it possible to detect carrier animals by collecting blood in ethylenediaminetetraacetate (EDTA) tubes and shipping the tubes to the laboratory on cold packs. Not enough is known about the regularity with which latently infected animals have virus in circulating white blood cells to predict the actual efficiency of the test in detecting carrier animals. However, APHIS is anxious to try the test on animals suspected of being infected with MCF virus. Anyone who wants to use this test for such animals is encouraged to contact the senior author at the address below.

(Dr. J. B. Katz, Dr. M. L. Frey, Diagnostic Virology Laboratory, National Veterinary Services Laboratories, APHIS, USDA, Ames, IA 50010, 515-239-8266)

Foot-and-Mouth Disease in Latin America

Foot-and-mouth disease (FMD) has been identified in Latin America since 1870, when the disease occurred simultaneously in Argentina, Brazil, Chile, and Uruguay, associated with the importation of cattle from Europe. Between 1870 and the 1970's, the pattern of disease outbreaks was characterized by periodic enzootics. These enzootics were attributable to inadequate zoosanitary control over the movement of livestock, inefficient inspection and quarantine of imported animals and products of animal origin, and, more recently, failure to carry out vaccination.

As a result of the devastating effects of the enzootics of the 1950's and the early 1960's on trade and cattle production, the governments of Latin America decided to intensify and systematize the control and eventual eradication of FMD by taking the following actions:

1. Strengthening the institutional infrastructure of the ministries of agriculture responsible for the prevention and control of animal diseases.

2. Creation of mechanisms to ensure national and regional coordination of efforts to control and eradicate animal diseases through harmonization of technology in campaign execution, efficient diagnosis of disease, development of methods to control information, and monitoring of vaccine production.

The development of these measures, which were of vital importance to the national programs for the control of FMD, was supported during the 1960's and 1970's by the Inter-American Development Bank, which loaned approximately $150 million (U.S.) to the countries involved. Technical assistance was provided to assist the veterinary services in the execution of the campaign from international agencies, including the Pan-American Health Organization and World Health Organization, the United Nations' Food and Agriculture Organization, and Inter-American Institute for Cooperation on Agriculture, as well as bilateral aid institutions, including USDA, Overseas Developmental Assistance, and the West German Agency for Technical Cooperation. These programs constituted the base for the creation and organization of the animal health services in most nations of South America.

In 1972, the South American Commission for the Control of FMD (COSALFA) was created by unanimous decision of the ministers of agriculture of Latin America at the fifth inter-American ministerial meeting for the control of animal disease and other zoonoses. COSALFA provides a forum where current technology can be discussed by representatives of the veterinary services of the nations of South America. The appropriate technology is then adopted in the execution of campaigns to control and eradicate vesicular disease. Since 1973, COSALFA has efficiently coordinated, promoted, and evaluated national and regional efforts directed at the eventual eradication of FMD.

By 1985, the nations of South America were making serious efforts to develop the infrastructure to fight the disease. All countries have now developed systems to monitor information related to animal disease, as well as services for epidemiologic surveillance, diagnostic laboratories, laboratories for the control of vaccines, governmental and private laboratories for vaccine production, and the national, regional, and zonal requirements for executing the campaign.

In most South American countries today, this infrastructure is utilized to combat not only FMD but all animal diseases of economic importance, including rabies, brucellosis, tuberculosis, and ectoparasitism.

Actual Situation

As a direct consequence of these actions, FMD has been eradicated from Chile, Argentina south of the 42d parallel, Guyana, French Guyana, and Surinam.

Though much remains to be done to eradicate FMD from the remainder of the continent, the annual incidence of the disease per 1,000 herds has decreased from 13–20 infected herds in the 1960's to 1 in 1990. Annual morbidity has declined from 200–300 per 1,000 animals to approximately 7 per 1,000 animals. From 1981 through 1990, 7,613 laboratory diagnoses of FMD were made in Argentina, Brazil, Chile, Paraguay, and Uruguay. Of that total, 30 percent were type O virus, 47 percent were type A, and the remaining 23 percent were type C. The following subtypes of virus were identified in 1990: O-1, A-81, A-24, and C-3. To control the disease, an inactivated trivalent A1(OH)3/saponin vaccine containing antigens prepared from O, A, and C types of the virus has been used for many years. Cattle are vaccinated every 4 months in order to ensure good immunity. An oil adjuvant vaccine is now being introduced and offers promise of providing longer immunity. It requires only annual vaccination in adult cattle and semiannual vaccination for young stock. This reduced schedule obviously substantially lowers the cost of assembling livestock.

In those areas now free of the disease—Chile, Guyana, French Guyana, Surinam, and Argentina south of the 42d parallel—official veterinary authorities immediately use rigorous measures to eliminate FMD when foci occur.

In spite of these efforts at a continental level, progress since 1987 toward eradication of the disease has remained static. The epidemic episodes described below in 1990 are cause for concern:

- The recurrence of FMD in Peru in the Departments of Ica, Junin, and Ayacucha after 14 years of freedom from disease;
- The increase of 33 percent in outbreaks in Ecuador;
- The increase of 50 percent in outbreaks in the Departments of Cochabama and Santa Cruz in Bolivia;
- The appearance of FMD in livestock in the Province of Rio Negro, which placed at risk cattle raised in the area of the Argentinean–Chilean border; and
- The transmission of the disease from animals located in the area of the Rio Salada to fattening cattle in the Provinces of Buenos Aires, La Pampa, Santa Fe, and Cordoba.

As mentioned earlier in this report, progress in the eradication of FMD has been almost nil during the last 5 years. The vast investment in the operation of veterinary services and production of vaccine, estimated to be at least $250 million (U.S.) per year, has not totally eliminated the disease or the associated physical losses. Therefore, zoosanitary regulations and trade restrictions involving livestock and animal products from South America continue to be enforced by those nations free of the disease. The failure to progress in the eradication of FMD is attributable to the following factors:

1. A general decline in the operation of official veterinary services due to insufficient funds.

2. Questionable vaccine coverage because livestock owners are permitted to administer vaccine themselves. (To correct this limitation, in the future cattle vaccination will be directly supervised by the veterinary services of each nation of South America.)

3. Failure in each country to implement selective and specific regional strategies to eradicate FMD that take into consideration the epidemiologic behavior of the disease in the predominant systems of cattle production.

4. Reduction in numbers of trained personnel, who are essential for all aspects of the campaign.

5. Difficulty of maintaining continuity in the execution and administration of the campaigns because of politically motivated interference from government officials outside the veterinary arena.

6. Failure to maintain adequate coordination and collaboration with other institutions involved in animal health, including universities, extension services, associations of livestock farmers, associations of veterinary surgeons, and cooperatives.

7. Failure to convince all members of society that they will benefit from the eradication of FMD.

8. Grave deficiencies in biologic security in private and public laboratories managing the virus, thus increasing the risk of escape of the virus from these establishments.

Actions Currently Being Adopted To Further the FMD Eradication Effort

Member countries of COSALFA, conscious of the problems already described and of the technological means available today for combating FMD, have introduced an updated program to eradicate the disease from the continent. To that end, they have held several international meetings to discuss and define policies and strategies for the work ahead. The first of these meetings was held in Buenos Aires in 1978.

Their actions were expanded in April 1987, when the Fifth Inter-American Meeting on Animal Health recommended that the Pan-American and World Health organizations and COSALFA prepare a hemispheric program for the eradication of FMD, including adequate measures for program implementation. Within this framework, during the late 1980's experts from each country formulated plans to eradicate the disease on a regional basis, dividing the continent into the following zones: Rio de la Plata basin (Argentina, Brazil, and Uruguay), the Andes (Bolivia, Peru, Ecuador, Colombia, and Venezuela), and Brazil and the Amazon area.

The regional project for the Rio de la Plata basin became a reality in July 1987, when authorities from Argentina, Brazil, and Uruguay signed a technical assistance agreement to harmonize strategies for the eradication of FMD. The agreement included actions to develop appropriate technology to improve diagnosis, implement a system of epidemiologic surveillance, administer the campaign efficiently, and train personnel. The total cost of the project is estimated to be $58.1 million (U.S.), which will be

partially covered by funds from loans granted to Argentina, Uruguay, and Brazil by the Inter-American Development Bank and the World Bank for the development of the nations' animal health services over the past 3 years.

The strategy to eradicate FMD in South America will therefore be based upon the following actions:

1. Regionalization, with the application of zoosanitary measures for control and eradication based upon the epidemiologic behavior of the disease. This strategy will take into consideration livestock production systems as well as the ecological, social, economic, political, and cultural conditions of the region. (One result of the regional approach to eradicating FMD is already encouraging: no outbreaks have been reported in Uruguay since May 1990. In Argentinean Mesopotamia, there has also been a decline in the number of outbreaks.)

2. Selection of areas where the disease will be eradicated, taking into account the administrative, technical, social, and economic capacity to execute the campaign.

3. Characterization of the ecosystems in accordance with the epidemiologic behavior of the disease.

Summary

Much work remains to be accomplished in order to eradicate FMD by the year 2005. Of particular importance are the strengthening of veterinary services, the education of farmers to encourage reporting of the disease, and the efficient execution of livestock vaccination.

(Clive B. Woodham, B.Vet.Med., M.Sc., M.R.C.V.S., Inter-American Development Bank, Washington, DC)

Focus on Rinderpest, an Update

A review of rinderpest and its recent history was presented in the fall 1988 issue (vol. 16, no. 3) of the Foreign Animal Disease Report. The present article describes the recent distribution of this disease and developments in its improved diagnosis and control.

Recent History

Africa. In the aftermath of the major epidemics of the early 1980's, the Pan-African Rinderpest Campaign (PARC), funded by the European Community and other donors and implemented through the Organization of African Unity, is leading a concerted vaccination campaign to rid the whole continent of this disease. In West Africa, no outbreaks of the disease have been reported since 1988. In East Africa, however, rinderpest has continued to be reported from Sudan, Ethiopia, Uganda, and Kenya. At present, endemic infection is established in that region. Fortunately, the virus has not spread into southern Kenya or Uganda to threaten economically important wildlife and cattle populations there and in neighboring countries.

In Uganda, Kenya, and Sudan, affected animals typically show fairly mild clinical signs, seldom exhibiting diarrhea or dying. Careful physical examination is needed to

detect fever and necrotic mouth lesions, which may be transient. In Ethiopia, more-virulent outbreaks of the disease have been seen in cattle of the central highlands and are attributed to contact with infected cattle being trekked to market from the lower rangelands, where the disease is reported to be of the mild, endemic type.

Middle and Near East. History repeats itself with outbreaks of rinderpest following close upon war and civil unrest. During 1990 and 1991, the disease was officially reported from Yemen, Oman, and Iran, and unconfirmed reports suggested its presence in Syria, Iraq, and Afghanistan. In the latter part of 1991, the disease appeared in eastern Turkey, from whence it rapidly spread several hundred kilometers west to threaten Europe. In so doing, it crossed with ease a wide *cordon sanitaire* intended to protect Europe from the far more easily transmissible virus of foot-and-mouth disease.

Europe. The first reported outbreak of rinderpest in Europe since the 1920's (omitting a contained outbreak in the Rome zoo in 1949) occurred in Georgia in 1990. Interestingly, the report noted that the disease affected only unvaccinated stock, posing the question as to why cattle in this region were still being vaccinated. With the epidemic outbreak in Turkey and the increasing administrative turbulence in the dissolving Soviet Union and its former satellites, the prospect of rinderpest outbreaks in Europe now seems higher than at any time since the Second World War.

Asia. Rinderpest remains endemic or sporadically epidemic in India and Sri Lanka and, although not reported, is probably present in neighboring countries. It is not reported in Southeast Asia. A recent outbreak in Mongolia, the first since 1935, suggests, however, that the virus may be more prevalent in Central and East Asia than the absence of reports would indicate.

International Control Campaigns. Rinderpest is rightly considered one of the most controllable of all diseases. Several international, donor-funded programs are now under way to assist individual countries in their control of the disease and to coordinate the efforts of these countries on a regional basis. PARC aims to control and eventually eradicate rinderpest throughout Africa. The South Asia Rinderpest Campaign has a similar objective in India and its neighbors, and an international campaign for West Asia is in preparation.

Technical Developments

Molecular Studies. As with most other viruses, recent research has focused on the genetic code of the virus with the aim of defining the genes and their products responsible for inducing protective immunity. One immediate benefit has been the development of recombinant vaccines (see below).

In-vitro differentiation of strains of rinderpest was previously impossible despite major differences in pathogenicity. However, recent sequencing of viral genes has begun to define strain differences that may provide a technique for the molecular mapping of isolates needed to assist the eradication campaigns.

Highly purified antigens derived from recombinant genes and monoclonal antibodies are being produced in sufficient quantity for use in the development of a new generation of more-sensitive diagnostic tests.

Diagnosis. The most extensively used diagnostic laboratory test is still agar gel immunodiffusion (AGID). Robust, easy, and uniquely specific, it is supplied in kit form

throughout Africa for use in the field and local laboratories. Its only drawback is its relative insensitivity for confirming disease in the mild cases so prevalent in areas where rinderpest is endemic. The collection of lymphoid tissues from alimentary tract lesions is not an option when dealing with owners who expect full recovery of their animals. Rapid agglutination tests using antibody-coated latex beads have shown their potential in laboratory experiments, and adaptations of the electrophoretic immunosorbent analysis (ELISA) on "dipsticks" or other small substrates should prove equally advantageous. Unfortunately, these rapid tests, which can be used beside the animal, have not reached the field and must surely benefit from further development using monoclonal antibodies and genetically engineered antigens. In these situations, conjunctival, nasal, or oral swabs can be used as a source of antigen. The former is particularly useful.

Rapid virus isolation has become a reality using continuously growing lines of bovine T lymphoblasts. Visible syncytia often develop in these cells after only overnight incubation with infected swab samples that are negative on the AGID test and that may require incubation for a fortnight before isolation on conventional cell monolayers.

Hybridization techniq5%s using cDNA probes have been successful in distinguishing between isolates of rinderpest and peste des petits ruminants virus but have not yet been refined for routine diagnosis. Of particular potential value is the development of a test employing the polymerase chain reaction to detect only minute fragments of viral RNA in tissues or swabs containing unviable virus and insufficient or decomposed viral antigens and RNA.

Serology. The indirect ELISA introduced for use throughout Africa in support of PARC was recently replaced by a competitive ELISA. The successor is said to give clearer demarcation between positive and negative sera, but as with its predecessor, it has not been widely used and will require further comparison with the widely accepted virus neutralization test.

Again, there is room for new tests, such as agglutination using genetically engineered antigens and a rapid virus neutralization test using bovine lymphoblasts.

Epidemiology. Cattle experiments have shown that certain moderately virulent strains of virus can cause abortion several weeks after clinical recovery of pregnant cows and that the aborted fetus may contain live virus. Transmission to cattle via contact was not achieved, but evidence indicates that quarantine periods for recovered pregnant cattle need to be reassessed. Other preliminary experiments have also shown that mild strains are not necessarily highly contagious. Such information is particularly useful for computer models recently developed in Africa and Europe, which are attempting to clarify the mechanisms that predispose to endemic infection and the ways in which control procedures, particularly vaccination, can most effectively prevent infection.

Vaccines. As mentioned above, recent molecular advances have resulted in the development of recombinant vaccines. *Vaccinia* recombinants expressing the F and/or H genes were developed independently in the United States, the United Kingdom, and Japan. These recombinant vaccines induce protective immunity in experimentally inoculated cattle and pigs and, because of the known thermostability of *Vaccinia*, offer great potential in hot climates. Veterinarians have remained cautious about the immediate adoption of the vaccines due to reservations about the widespread use of *Vaccinia* and the lack of data on the length of the protective response. A recombinant

using a less pathogenic variant of *Vaccinia* has now been produced in the United States and may be more acceptable for use in the field. Clinical trials should soon assess the length of the immune response it induces.

Another program in the United States has resulted in significant improvements to the keeping qualities of the commonly used cell-culture attenuated Kabete "O" vaccine. Growth of the virus in Vero cells gave much higher prefreeze-drying titers, and a more intensive lyophilization schedule produced lower residual moisture content. The result is a lyophilized vaccine that can be kept without refrigeration at high ambient temperatures for up to 2 months, as has been successfully confirmed during field trials in Niger.

PARC also commissioned the development in France of a clone of attenuated Kabete "O" vaccine that was significantly more thermostable than the basic vaccine. PARC has now funded a program to combine these two techniques, the French thermostable clone and the United States' improved lyophilization of Vero-grown virus. The resultant product, to be named Thermovax, should be highly thermostable and as inexpensive, safe, and immunogenic as its Kabete "O" parent stock.

Research Needed. Some of the research issues raised in the 1988 article in this report still remain appropriate objectives today. Effective techniques for the differentiation of strains of rinderpest virus and a serologic test to distinguish vaccinated animals from those previously infected with field virus are still required. The latter issue will be resolved if the recombinant pox vaccines are universally adopted. Alternatively, with the current development of Thermovax, now might be the time to insert or delete a genetic marker that allows a distinctive antibody development in vaccinates.

Now that the thermostability of freeze-dried vaccine has been improved, an attempt to increase the stability of the reconstituted vaccine would, if successful, result in more cattle being inoculated with live virus.

Research into the mechanism determining virulence and its attenuation in endemic areas is now under way and should be assisted with research grants from international control campaigns.

One new research need is the development of more-sensitive diagnostic tests. The previous article did not specify this as necessary, but recent experience with mild strains in East Africa has clearly demonstrated the shortcomings of AGID and the need for sensitive and simple new tests.

Other Problems. The essential tools for the eradication of rinderpest—a way to control the movement of animals and an effective, cheap vaccine—have been available for 30 years in Africa and India, but the disease is proving difficult to eradicate. Recent successes in West Africa show how effective the tools are, but the virus still persists in East Africa. Why? Responsible authorities have been tempted to excuse their failures by suggesting inherent differences in the strains of virus persisting in their region. There is no evidence to support this; the most likely reason is that East Africa has a far greater number of cattle than West Africa, and that throughout much of the region the control of animal movement has become haphazard. In addition, vaccination campaigns are not always pursued with sufficient diligence, or in synchrony with campaigns across contiguous national boundaries, or with the fullest support of the cattle owners. When these problems are solved, then rinderpest will be eradicated in East Africa.

In parts of Asia where rinderpest is also independently established in sheep and goats, ineffective control of animal movement contributes greatly to its persistence. An additional factor there has been the continuing use of poor-quality or outdated vaccines and reluctance to adopt new ones. Hopefully, education and current international campaigns will help to rectify this.

New morbilliviruses have recently been found in pinnipeds and cetaceans, prompting speculation about the origins of the viruses. Were they long-standing infections of their respective host populations that have only now been detected following "green" public interest in "die-offs," or have they recently evolved after chance infection with established plagues such as rinderpest or distemper?

The clear biochemical differentiation of the African from the Asian strains of peste des petits ruminants virus has led one authority to speculate that it might conceivably have evolved on at least two separate occasions, presumably from rinderpest. Should this be so, then, considering the unknown origins of the new marine plagues and the recent detection of peste des petits ruminants in India, the permanent eradication of rinderpest might conceivably require the eradication of that disease as well. However, with the exception in West Africa, rinderpest is currently experiencing a resurgence, and such a dilemma is still several years in the future.

(Dr. C. M. Groocock, Veterinary Attache, International Services, APHIS, USDA, Hyattsville, MD 20782, 301-436-8892)

Clarification

It was stated on page 4 of the Fall 1991 issue of the Foreign Animal Disease Report that "The Pan-American Foot-and-Mouth Disease Center, Rio de Janeiro, Brazil, diagnosed vesicular stomatitis New Jersey in El Salvador and Honduras for October and November 1990 and in Nicaragua and Costa Rica during October 1990."

To clarify: Laboratory samples from Central America, Belize, and Panama to be tested for vesicular diseases are submitted to the Laboratorio de Diagnostico de Enfermedades Vesiculares (LADIVES), a USDA support lab in Panama. The results of these tests are then sent to the Pan-American Foot-and-Mouth Disease Center, Rio de Janeiro, Brazil, where they are distributed in a monthly bulletin to the OIE.

The Foreign Animal Disease Report's former editor, Edwin I. Pilchard, noticed that vol. 19, no. 4 was incorrectly labeled "Winter 1992." It should have been called the Winter 1991 issue.

Subject Index

A subject index covering articles that appeared in volumes 10 through 19 of the Foreign Animal Disease Report is available upon request.

Questions about the FAD Report may be sent to:

Dr. M. A. Mixson, Editor
USDA, APHIS, VS
Room 741, Federal Building
6505 Belcrest Road
Hyattsville, MD 20782

Thirty days before moving, send address change and, if possible, mailing label from latest issue to:

Distribution Management Section
USDA, APHIS, MSD, PDMB
Room G-110, Federal Building
6505 Belcrest Road
Hyattsville, MD 20782